Everything being in readiness for the operation, she desired her clergyman to offer prayer. She then bade us all "good-bye," and commenced the inhalation of ether as calmly as she would ordinarily have sat down to rest. No untoward symptoms occurred during the operation. The artery was readily compressed over the pubes, and very little hemorrhage occurred. The circular integumental operation was the one chosen, and she passed through it without an unfavourable symptom, was comfortably put in bed, partial consciousness returned for half an hour, when a sudden pallor came over her countenance and she passed away calmly.

I am indebted to my father, Dr. Isaac Smith, of Foxboro', Mass., and to Drs. John B. Whitaker, Charles Hayes, and A. J. Sullivan, of this city, for their highly valuable assistance during the amputation; also to Dr. J. C. Warren, of Boston, Mass., a highly accomplished microscopist, for the following pathological statement in regard to specimens of the diseased limb which I furnished for examination. He says he finds the—

"Cutis vera and subcutaneous cellular tissue to be very much thickened by a growth of dense fibrous tissue which seems to take its rise from the *outer coats* of the bloodvessels, which are very numerous and large. There are, also, vessels which appear to me to be *enlarged lymphatics*. It is not possible to distinguish the boundary line between the cutis and the parts beneath it. Most of its organs appear to be destroyed. In the specimens examined there were no traces of hair follicles or sebaceous glands. Traces of sweat glands were found here and there, and in one instance a sweat duct was found entire. The papillæ were much larger than in normal skin."

I believe that although this case was fatal that other cases might give a more hopeful prognosis. The amputation cleared the diseased parts entirely, and the only solution of the cause of dissolution seems to be that it was the result of shock. The limb weighed a trifle over *one hundred* pounds avoirdupois.

FALL RIVER, MASS., March 14, 1870.

ART. XIII.—*Pathology of Alopecia Areata.* By L. A. DUHRING, M.D., of Philadelphia.

FOR the last twenty years micrographers and dermatologists have been discussing whether alopecia areata is due to the presence of a fungus or other causes. This question is worthy of investigation in order that we may be able to treat the disease in a rational manner.

Alopecia areata is a disease confined to the hair system, generally met with upon the scalp, and characterized by whitish bald patches occurring in one or more parts, various in shape and size, but generally circular. This affection must not be confounded with tinea tonsurans, which it often closely resembles, for the latter exists under totally different conditions from those present in alopecia areata, and a distinction must be drawn between them. Passing over the early literature of this affection, Gruby, in 1843, declared that he had discovered a fungus in this disease, to which he gave the name of *microsporon audouini.* Shortly after this many researches and experiments were made in quest of this fungus, some corroborative of the statement of Gruby, others contradictory of it. The discussion has continued to the present day, and the only means of solving the problem is by aid of the microscope. The fact that this affection bears a resemblance in its seat and appearance to tinea tonsurans should not influence us in our investigation. The question to be solved is: When we examine the hairs and epidermis properly do we see a fungus or not? This appears like an easy problem to solve; at the same time it is but just to remark that the investigation requires a certain amount of skill and practice to avoid error.

The upholders of the present day of the parasitic nature of alopecia areata, foremost amongst whom are Tilbury Fox, Bazin, and Hardy, contend that the disease is produced by the presence of the microsporon audouini, and that this is the only cause of the disorder. This fungus is represented as consisting of mycelium and spores, the former being more abundant than in the trichophyton, while the latter are smaller and less numerous, and have all the general properties of the other fungi, and are found and demonstrated in the same manner as the trichophyton and achorion Schœnleinii. Though the object of the present remarks is to refer particularly to the nature and cause of the disease under consideration, yet, perhaps, a few preliminary words in regard to the mode of examination of the hairs will not be out of place. Among the many reagents we shall only refer to three: solution of potassa, glycerine, and water, and it is proper all three should be employed for a thorough examination, though when in search of a suspected fungus it is preferable to use the first-named solution of the following strength: Potassa fusa twenty-five grains, aqua destillata one ounce. Potassa has a definite action upon the epidermic tissue, breaking up and dissolving the adhesions of the cells at once, while after contact for some time it completely destroys the cell structure, the destructive action varying according to the strength of the solution used. Applied for immediate use of the above strength it merely separates and frees the cells from one another, without acting injuriously upon the epidermic cells of the hair itself to such an extent as to render its employment objectionable. At the same time a solution of potassa has no determined destructive action upon vegetable parasites; on the contrary, its use is always indicated in the search for these delicate structures, since their characters are more plainly brought out by its breaking up the bed of epidermic tissue, thus leading to their detection, when otherwise this would be extremely difficult, if not impossible. In order to get rid of superfluous fatty matter a few drops of sulphuric ether should be used, though this will by no means clear the hair and field completely from fat and sebum. For the purpose of obtaining a proper view of the hair, about five hundred diameters magnifying power is required, and it is well even to use higher powers occasionally to determine doubtful points; moreover a strong light is necessary to penetrate the substance of the hair.

Alopecia areata presents different appearances in its various stages, but if a patch be examined when the disease first shows itself, the hairs are seen to be fewer in number than normal, and diminished in bulk, intermingled with little, short, broken-off hairs scattered here and there over the patch. In the course of a week or two all these will have disappeared, and a perfectly bald spot with a clearly-defined margin of apparently sound hairs will be seen remaining. It is in these short, stumpy, broken-off hairs that we might expect to find evidence of disease, or at least the most marked alteration. These broken-off hairs, when extracted with the forceps, come out of their sheath without any resistance, and when placed on a glass slip with a drop of solution of potassa covered with a thin glass, and placed under the microscope, are seen terminating abruptly in a pear or club-shaped extremity, instead of the thick, long, spongy, luxuriant-looking bulb with its well-defined, transverse striæ, seen in normal hairs plucked from the scalp. The bulb is contracted, shrivelled, and atrophied, and surrounded with only a scanty supply of sebum and epidermic cells. Its periphery is generally sharply defined with a zigzag border, or in other cases presents a rounded appearance. The root retains its normal look with the exception of being diminished in size. In the shaft, however, we notice, as we approach the free end, an even and gradual distension, terminating generally in an oval swelling or bulging close to the end of the hair, and then suddenly tapering down, finishing in a broken, ragged extremity. These are the appearances we invariably find in a marked case of alopecia areata, *with no sign of fungus!*

When the hairs on the patch that have not been broken, or such as are diseased and are just ready to break, are examined, we see the same atrophied, shrunken bulb, the same feeble root, and the shaft increasing in diameter towards the free end. Many of them escape being injured and broken, and, having been detached from their papilla, act as a foreign substance, and are rapidly cast off.

Is the shrunken, atrophied condition of the bulb characteristic of this disease, or is it found elsewhere? If a hair that has fallen naturally from a healthy head be examined, almost exactly the same appearance of the bulb is seen; it is shrivelled and dead. This atrophied bulb is also found in hairs that have lived their normal life, and, having been detached from their papilla, are thrown off; in one case it is health and in the other disease. Instead of the normal growth and death of the hair, we have a sudden arrest of nutrition from some cause, and a rapid wasting and death in consequence, due most probably to atrophy of the papilla itself. If there be a defective nutrition in the papilla, a paralyzation of nerve force, the bulb will suffer first, but the life of the whole hair is quickly affected, it withers, dies, and is cast away with its blighted bulb and shaft.

Owing to some cause or other yet unknown this temporary loss of vitality in a part occurs, and there is alopecia areata, in some cases to a slight extent, in others involving the whole hair system, scalp, axillæ, and pubes; thus the entire body is deprived temporarily of its hair.

Such in a few words seems to be the most rational and probable explanation of the loss of hair in the disease under discussion, namely, a sudden arrest of nutrition in the papilla. The bulging or oval distension of the shaft at its end, referred to, due, as some consider, to the presence of fungus, is easily explained as follows: The shaft not receiving its proper nourishment from the papilla, its extreme end suffers most, and does not thrive; the filaments not being sustained as usual and losing their vitality, tend to separate and disintegrate, thus stretching the epidermic membrane and causing the appearance described. The fact that the filaments do separate is clearly demonstrated with a high power and a strong, penetrable light; moreover this distension to a greater or less extent is constant, and in the same position in every case.

When the devitalized and brittle hairs are subjected to violence, as from a comb or brush, they are broken off at the point where there is the greatest force exerted, just where they emerge from the scalp, leaving the jagged end, a small portion of the shaft, the root and bulb. In other words, nothing but the stumpy, broken hairs remain in the scalp; these soon loosen from their papilla, separate from the sheath, and are cast off a dead substance. If the hair breaks because it is greatly distended and brittle, it might be expected to break at the bulging, the point of its greatest distension, and instead of a tapering end, a wide-spread, bushy extremity be found. The hair undoubtedly does break at the point of greatest distension, but as soon as this happens the epidermic membrane, which gives contour to the hair, contracts and holds together the free filaments; thus acting as an elastic membrane and not allowing the broken filaments to spread themselves.

It may be asked: How does it happen that a fungus should have been so minutely described, if none actually exists? About the roots and shafts of all hairs normal or diseased, varying with the case, there exists an appreciable amount of sebum, broken-up epidermic cells, and débris. When subjected to a reagent, sebum has a tendency to split and break up into fine particles, and these often adhere so closely and with such tenacity to the hairs as to accurately resemble spores.

I cannot help thinking that it is to these deceptive little particles of sebum that the name of microsporon audouini owes its existence!

Upon two occasions the supposed fungus, the microsporon audouini, was demonstrated to me by experienced and well-known dermatologists; in both cases I had the satisfaction of proving conclusively by means of reagents, that the regularly divided, rounded, bright little bodies were simply aggregations of sebum.

I may add, moreover, that I have found this curious and spore-like arrangement of sebum upon hairs, normal in every respect. In conclusion, let it be borne in mind that the foregoing observations apply exclusively to the short, broken-off hairs which are found in the earliest recognizable stage, the typical hairs of alopecia areata; after this period the disease presents a different appearance, namely, complete baldness, with an edge of apparently normal hairs, which continues until recovery begins.

ART. XIV.—*Bromides in Epilepsy.* By BENJAMIN SWAN, M.D., of San Francisco, California.

IN 1867-8 I was one of the resident assistants to Dr. Whittelsey, at the Nursery Hospital, Randall's Island, New York city, and among the inmates were some fifty epileptics. The bromides were used freely, and the result of my year's experience was that as long as the system was kept saturated with the bromide "the fits" would be prevented; but an omission of the medicine, or failure to increase the dose after a time, involved a return of the disease.

A boy was brought to the Island who had epilepsy, averaging five attacks per week. He was put on Dr. Brown-Séquard's formula: ℞.—Ammon. bromid. ℈ij ; Potassii bromid. ʒvj ; Potass. bicarb. gr. xvj ; Tr. Calumbo, ʒjss; Aquæ ad. ℥iv.—M. Cap. ʒj t.i.d. The attacks became less frequent. In course of a few weeks, after an interval of nearly a month, they began to increase in frequency again, when the dose was increased.

After this, whenever the attacks grew more frequent, the dose was increased, so that in May, 1867, he was taking drachm doses of the bromide of potassium, and a proportionate dose of bromide of ammonium ; on this he went from May till August without an attack.

Great attention was paid at the same time to the bowels, and a purge of calomel and podophyllin was given occasionally. In the latter part of August his father took him from the Island, contrary to my advice, as he could not afford to buy the bromides, saying the boy was well, as "he'd had no fits for four months," and "he'd risk it."

In the middle of October he was brought back, having fits at the rate of three a week. The treatment resumed diminished the frequency of the attacks, but the most heroic doses failed to produce an interval longer than three weeks.

In another case a boy, after reaching drachm doses, had large ulcers appear on his legs. The dose was diminished, when the ulcers healed and the fits returned. We again increased the dose, the "fits" disappeared and the ulcers reappeared.

I might give numbers of cases, all seeming to prove that a point is finally reached when the system becomes insensible to the action of the medicine.

I could not therefore call a case cured while still under the influence of the medicine, and, judging from my own experience, I should very much fear to reduce the dose; and, on the contrary, expect to be obliged to *increase* it.

I believe the bromides to be the best remedies in epilepsy, but I have yet to hear of a case that was perfectly cured.